Flame Retardant Alternatives

Proprietary J: Aryl phosphate

Hazard Review

Proprietary J: Aryl phosphate
Existing Data Summary Table – Human Health Endpoints

✓= Endpoint characterized by existing data * = Data available but not adequate ✘ = Endpoint not applicable

As noted in this key, a check mark indicates that an endpoint was adequately characterized by existing studies. It does not indicate a positive or negative result for that particular endpoint.

Acute Toxicity	
Oral	✓
Dermal	✓
Inhalation	*
Eye irritation	✓
Dermal irritation	✓
Skin sensitization	
Subchronic Toxicity	
28-Day oral	*
90-Day oral	*
Combined repeated dose with reproduction/ developmental toxicity screen	
21/28-Day dermal	*
90-Day dermal	
90-Day inhalation	
Reproductive Toxicity	
Reproduction/ developmental toxicity screen	
Combined repeated dose with reproduction/ developmental toxicity screen	
Reproduction and fertility effects	

Developmental Toxicity	
Reproduction/ developmental toxicity screen	
Combined repeated dose with reproduction/ developmental toxicity screen	
Prenatal developmental	*
Chronic Toxicity	
Chronic toxicity (two species)	
Combined chronic toxicity/ carcinogenicity	
Carcinogenicity	
Carcinogenicity (rat and mouse)	
Combined chronic toxicity/ carcinogenicity	

Neurotoxicity	
Acute and 28-day delayed neurotoxicity of organophosphorus substances (hen)	✓
Neurotoxicity screening battery (adult)	
Developmental neurotoxicity	
Additional neurotoxicity studies	
Immunotoxicity	
Immunotoxicity	
Genotoxicity	
Gene mutation in vitro	*
Gene mutation in vivo	
Chromosomal aberrations in vitro	*
Chromosomal aberrations in vivo	
DNA damage and repair	
Other	*

Proprietary J: Aryl phosphate
Existing Data Summary Table – Properties, Fate, and Ecotoxicity

✓= Endpoint characterized by existing data * = Data available but not adequate ✘ = Endpoint not applicable

As noted in this key, a check mark indicates that an endpoint was adequately characterized by existing studies. It does not indicate a positive or negative result for that particular endpoint.

P/Chem Properties	
Water solubility	✓
Octanol/water partition coefficient	✓
Oxidation/reduction	
Melting point	✓
Boiling point	✓
Vapor pressure	✓
Odor	
Oxidation/reduction chemical incompatibility	
Flammability	
Explosivity	
Corrosion characteristics	
pH	
UV/visible absorption	
Viscosity	✓
Density/relative density/bulk density	✓
Dissociation constant in water	
Henry's Law constant	✓

Environmental Fate	
Bioconcentration	
Fish	*
Daphnids	
Green algae	
Oysters	
Earthworms	
Metabolism in fish	
Degradation and Transport	
Photolysis, atmosphere	
Photolysis, water	✓
Photolysis in soil	
Aerobic biodegradation	*
Anaerobic biodegradation	
Porous pot test	
Pyrolysis	
Hydrolysis as a function of pH	
Sediment/water biodegradation	✓
Soil biodegradation w/ product identification	
Indirect photolysis in water	
Sediment/soil adsorption/desorption	*

Ecotoxicity	
Aquatic Toxicity	
Fish acute LC50	*
Daphnia acute EC50	*
Mysid shrimp acute LC50	
Green algae EC50, NOAEC, LOAEC	
Fish chronic NOAEC, LOAEC	
Daphnia chronic NOAEC, LOAEC	
Mysid shrimp chronic NOAEC, LOAEC	
Terrestrial Organism Toxicity	
Bird LD50 (two species)	
Bird LC50 (two species)	
Bird reproduction	
Earthworm EC50, LC50, NOAEC, LOAEC	

Chemical Identity

Proprietary J: Aryl phosphate
Synonyms
CAS
MF
MW
SMILES

Omitted from this report are a number of studies conducted on [Formulation 2] (~43% Proprietary J). The omitted studies included 21-day dermal toxicity, 90-day aerosol inhalation toxicity, and 90-day oral (feeding) toxicity in rats and neurotoxicity studies in hens. Replacement studies subsequently commissioned by the contracting company are reviewed here.
Many health effects studies have been conducted on commercial products that are mixtures of Proprietary J and closely related compounds: [Chemical 1], [Chemical 2], and [Chemical 3]. Typical composition data for these products are given in Table 13-1 below.

Table 13-1. Composition data (%) for selected t-butylated aryl phosphate products

Component	[Formulation 1][a]	[Formulation 2][b]	[Formulation 3][c]	[Formulation 4][d]	[Formulation 5][e]
Proprietary J	43	43	>99	30-35	73
[Chemical Class 1]	23				
[Chemical 1]		14		30-35	
[Chemical 2]		2		10-15	
[Chemical 3]	34	40		15-25	27
stabilizers			<1		

[a]Ref. 15
[b]Ref. 42
[c]Ref. 7
[d]Ref. 22
[e]Ref. 63. Ref. 58 reported that [Formulation 5] was [Chemical Class 1], but did not report the precise concentration of Proprietary J. After saponification, the isomer distribution of the [Chemical Class 2] portion was 39.2% [Chemical 4] and [Chemical 5] and 59.7% [Chemical 6].

Human Health Endpoints

ACUTE TOXICITY

Acute Oral Toxicity (OPPTS Harmonized Guideline 870.1100; OECD Guidelines 425, 420, 423, 401).

Conclusion:

The available acute oral toxicity data were judged adequate to meet the endpoint.

Basis for Conclusion:

Despite reporting deficiencies in some studies (lack of precise information on substance purity, group size), the available data indicate acute oral LD50 values exceeding 5,000 mg/kg for [Chemical Class 1] tested by methods equivalent to guidelines.

Critical Studies:

Type: Acute oral toxicity
Species, strain, sex, number: Rat, CD Sprague-Dawley, 5/sex
Doses: 5,000 mg/kg
Purity: isomeric mixture of [Chemical Class 1] as [Formulation 7] (Ref. 7)
Vehicle: None
Method: Based on OECD Guideline 401. Rats examined for mortality and clinical signs frequently on day 1, twice daily thereafter to day 14. Body weights recorded on days 1, 8, and 15. Gross necropsy on all rats.
Results: No deaths. Clinical signs in all rats after dosing included pilo-erection, hunched posture, and abnormal gait (waddling). Half of the animals had diarrhea. Clinical signs resolved by day 8. There were no effects on body weight gain or terminal necropsy findings. Acute oral LD50 was greater than 5,000 mg/kg in rats.
Reference: Ref. 25

Type: Acute oral toxicity
Species, strain, sex, number: Rat, Sprague-Dawley, 3/sex
Dose: 5,000 mg/kg
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 10]. Ref. 20 reports composition of 60-100% [Chemical Class 1] and 7-13% [Chemical 3].
Vehicle: None
Observation period: 14 days
Method: Rats observed 14 days after single dose, gross necropsy on all rats. Body weights recorded days 0, 7, and 14.

Results: No deaths. Clinical signs included abdominogenital staining, chromorhinorrhea, and decreased locomotion, all subsiding by day 10. All rats gained weight. No gross lesions. The LD50 exceeded 5,000 mg/kg.
Comment: This study was equivalent to a limit test under OPPTS 870.1100 except that the group size was 3/sex rather than 5/sex.
Reference: Ref. 16

Type: Acute oral toxicity
Species, strain, sex, number: Rat, Sprague-Dawley, males and females, numbers not reported
Dose: 15,800 mg/kg
Purity: Near pure Proprietary J
Vehicle: None
Observation period: 14 days
Method: Rats observed 14 days after single dose.
Results: The LD50 exceeded 15,800 mg/kg in rats (specific mortality results were not reported).
Reference: Ref. 34

Additional Studies and Information:

Ref. 10 evaluated the oral toxicity of [Chemical Class 1] administered to rabbits by oral gavage in 8% gum acacia; rabbits were observed "several days" for clinical signs of toxicity. There were no effects in single rabbits receiving 1,000 or 3,000 mg/kg of Proprietary J or [Chemical 1], or 1,000 mg/kg of [Chemical 2].

In another study, male rabbits were administered [Chemical Class 1] by oral gavage in 5% gum acacia and observed for varying amounts of time (Ref. 11). One rabbit received 2,000 mg/kg of Proprietary J and was observed for 5 days and two rabbits received 5,000 mg/kg and were observed for 4 or 16 days. All showed hepatic degeneration and two had kidney effects (tubular degeneration and congestion or swelling); necrosis of the stomach (high dose) and moderate lung congestion (low dose) were seen in single animals. A parallel experiment with rabbits treated with [Chemical 1] had similar results: liver effects in 1/1 at 2,000 mg/kg (killed day 5) and 2/2 at 5,000 mg/kg (killed day 4 or 17), and lung congestion and cloudy swelling of the kidneys in one high- and one low-dose animal. No deaths and no overt clinical signs were seen in rabbits treated with either compound.

A related compound, [Chemical 2] (containing 1-2% [Chemical 5]), was administered at doses of 3,000 or 10,000 mg/kg by oral gavage in olive oil to rats (Ref. 12). Mortality was 1/5 at the low dose and 1/3 at the high dose, but the length of the observation period was not reported.

As described in a robust summary, there were no deaths and no gross lesions in Sprague-Dawley rats (5/sex) orally exposed to 5,000 mg/kg [Formulation 5] (see Note e in Table 1) and observed for 14 days (Ref. 53 cited in Ref. 1). Clinical signs (depression, diarrhea, and stains on fur and around the nose) resolved by day 6. A letter from Ref. 52 to EPA suggests that this study was conducted under the 1978 EPA proposed test guidelines.

Acute Dermal Toxicity (OPPTS Harmonized Guideline 870.1200; OECD Guideline 402)

Conclusion:

The available acute dermal toxicity data were judged adequate to meet the endpoint.

***Basis for Conclusion*:**

Despite reporting deficiencies in some studies (lack of precise information on substance purity, group size), the available data indicate acute dermal LD50 values exceeding 2,000 mg/kg for Proprietary J tested by methods equivalent to guidelines.

Critical Studies

Type: Acute (24-hour) dermal toxicity
Species, strain, sex, number: Rat, Sprague-Dawley, 3/sex
Dose: 2,000 mg/kg
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 10]
Vehicle: None
Exposure period: 24 hours
Method: Undiluted test material applied to intact clipped dorsal skin. Treated areas occluded, washed after 24 hours with methanol and then water. Animals observed for 3 hours after dosing and daily thereafter for 14 days. Body weights recorded on days 0, 7, and 14. All subjected to gross necropsy.
Results: No deaths, clinical signs, local irritation of the application site, or gross necropsy lesions. All rats gained weight. The acute dermal LD50 exceeded 2,000 mg/kg in rats.
Comment: This study was equivalent to a limit test under OPPTS 870.1200 except that the group size was 3/sex rather than 5/sex.
Reference: Ref. 17

Type: Acute (24-hour) dermal toxicity
Species, strain, sex, number: Rabbit, New Zealand albino, male and female, numbers not reported
Dose: 7,900 mg/kg
Purity: near pure Proprietary J
Vehicle: None
Exposure period: 24 hours
Method: Undiluted test material applied to intact clipped dorsal skin. Treated areas occluded, washed (liquid unspecified) after 24 hours. Animals held for 14 days after which all subjected to gross necropsy.
Results: The acute dermal LD50 exceeded 7,900 mg/kg. The study did not report necropsy findings or specific mortality results.
Reference: Ref. 34

As described in a robust summary, mortality was 1/10 among New Zealand White rabbits (5/sex) that received a dose of 2,000 mg/kg [Formulation 5] (see Note e in Table 1) on intact and abraded skin and were observed for 14 days (Ref. 54 cited in Ref. 1). Clinical signs included depression and mild diarrhea. No gross lesions were observed at necropsy. A letter from Ref. 52 to EPA suggests that this study was conducted under 1978 EPA proposed test guidelines.

Acute Inhalation Toxicity (OPPTS Harmonized Guideline 870.1300; OECD Guideline 403)

Conclusion:

The available acute inhalation toxicity data were judged inadequate to meet the endpoint.

Basis for Conclusion:

The only relevant data were for a study available only as a robust summary and for which the Proprietary J content was uncertain.

As described in a robust summary, no mortality and no body weight effects were observed among Sprague-Dawley rats (5/sex) that were exposed for 4 hours to an aerosol of [Formulation 5] (see Note e in Table 1) at the highest attainable concentration, 3.1 mg/L (Ref. 54 cited in Ref. 1); the particle size distribution of 2.5-2.8 μm suggests that the particles were respirable. Ruffled fur was the only clinical sign observed over a period of 14 days. The only gross lesions observed were lung effects (reddened or whitish coloration) in two females.

Acute Eye Irritation (OPPTS Harmonized Guideline 870.2400; OECD Guideline 405)

Conclusion:

The available eye irritation data were judged adequate to meet the endpoint.

Basis for Conclusion:

Despite some uncertainty as to the purity of test substances, the available studies used methods equivalent to the guidelines and agreed that Proprietary J was not an eye irritant.

Type: Acute eye irritation
Species, strain, sex, number: Rabbit, New Zealand White, 3 (sex not reported)
Doses: 0.1 mL
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 7]
Vehicle: None
Method: Cites OECD Guideline 405. Eyes examined after 1 hour, then 1, 2, 3, 4, and 7 days.
Results: No positive responses; no damage to cornea or iris. Mild conjunctival inflammation was observed in 3/3 animals 1 hour after instillation only. All normal by 24 hours.
Reference: Ref. 27

Type: Acute (4-hour) eye irritation
Species, strain, sex, number: Rabbit, New Zealand White, 3 females
Doses: 0.1 mL
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 10]. Ref. 20 reports composition of 60-100% [Chemical Class 1] and 7-13% [Chemical 3].
Vehicle: None
Method: Instilled into eye. Eyes assessed via Draize method at 1, 24, 48, and 72 hours.
Results: No eye irritation was observed at any timepoint.
Comment: Although the study was designated "non-definitive", it was consistent with the OPPTS guideline.
Reference: Ref. 18

Additional information

As described in a robust summary, 0.1 mL [Formulation 5] (see Note e in Table 1) was a mild ocular irritant to rabbits (Ref. 56 cited in Ref. 1). Mild redness of the conjunctiva persisted to 24 hours in 2/9 and to 48 hours in 1/9, but was resolved by 72 hours. The eye irritation observed in this study may reflect compositional differences between [Formulation 5] and the other Proprietary J materials tested.

Acute Dermal Irritation (OPPTS Harmonized Guideline 870.2500; OECD Guideline 404)

Conclusion:

The available dermal irritation data were judged adequate to meet the endpoint.

Basis for Conclusion:

Despite some uncertainty as to the purity of test substances, the available studies used methods equivalent to the guidelines. Results indicated no or mild dermal irritation.

Critical Studies:

Type: Acute (4-hour) dermal irritation
Species, strain, sex, number: Rabbit, New Zealand White, 3
Doses: 0.5 mL
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 7] (relationship to the composition of [Formulation 3] reported in Table 1 is not known)
Vehicle: None
Method: Followed OECD Guideline 404. Hair clipped. Material applied for 4 hours to approximately 10-cm square area on back, semi-occlusive dressing. Site rinsed with water, examined after 30 minutes and days 2, 3, and 4; additional observations on days 5 through 11.
Results: Very slight or well-defined erythema with or without very slight edema was seen in two animals immediately, persisting though day 8 and day 10. Very slight erythema without edema in

third animal on days 2 and 3 only. A 4-hour exposure to the test material elicited mild reversible irritation.
Reference: Ref. 26

Type: Acute (4-hour) dermal irritation
Species, strain, sex, number: Rabbit, New Zealand White, 2 males and 1 female
Doses: 0.5 mL
Purity: Not reported; isomeric mixture of Proprietary J as [Formulation 10]. Ref. 20 reports composition of 60-100% [Chemical Class 1] and 7-13% [Chemical 3].
Vehicle: None
Method: Test material applied to clipped, intact skin and occluded. After 4 hours, sites were wiped clean with methanol and rinsed with tap water. Scoring for irritation was done 30 minutes after wiping and then daily for 3 days. Clinical signs were observed.
Results: No signs of irritation (erythema or edema) were noted at any timepoint. The primary irritation index was 0/8.0; the material was non-irritating to intact rabbit skin
Reference: Ref. 19

As described in a robust summary, [Formulation 5] (see Note e in Table 1) was a mild dermal irritant to rabbits, yielding a primary irritation score of 0.50 (Ref. 57 cited in Ref. 1).

Skin Sensitization (OPPTS Harmonized Guideline 870.2600; OECD Guideline 429)

Conclusion:

No available skin sensitization data.

Basis for Conclusions:

No pertinent studies were located that followed or were similar to the guideline listed above, or were otherwise relevant to skin sensitization.

SUBCHRONIC TOXICITY

Subchronic Oral Toxicity (28-day, 90-day, or combined with reproductive/developmental)

Conclusion:

The available subchronic oral toxicity data were judged marginally adequate to meet the endpoint.

Basis for Conclusion:

A 90-day oral toxicity assay on 73% [Formulation 11] was similar to guidelines except for the lack of testing for ophthalmological effects and neurological function and that the high dose was significantly lower than the limit dose. The study did identify target organs that might show

histopathology at higher exposure levels. The Proprietary J content of tested materials was less than 50% in a 1-month assay.

- **Repeated Dose 28-Day Oral Toxicity in Rodents (OPPTS Harmonized Guideline 870.3050; OECD Guideline 407)**

Ref. 42 exposed groups of Sprague-Dawley rats (10/sex/group) to diets providing [Formulation 2] (43% Proprietary J, see Table 1) at target doses of 0, 250, 500, 750, 1,000, or 2,000 mg/kg/day (nominal doses of 213, 442, 660, 898, and 1,710 for males and 234, 454, 690, 898, and 1,867 for females) for 1 month. Reduced food consumption was observed in males at 2,000 mg/kg/day; reduced body weight gain in males at ≥750 mg/kg/day and females at 2,000 mg/kg/day. There were no deaths, but hepatic enlargement was noted in all groups in a dose-related fashion; discoloration of kidneys was observed in male rats at ≥500 mg/kg/day. The lowest dose was a LOAEL; methodological limitations of the study include the lack of examinations for histopathology, hematology, or clinical chemistry.

- **90-Day Oral Toxicity in Rodents (OPPTS Harmonized Guideline 870.3100; OECD Guideline 408)**

Type: 90-day oral (diet) toxicity in rodents
Species, strain, sex, number: Rat, Sprague-Dawley (20/sex/group)
Doses: 0, 100, 400, or 1,600 ppm [Formulation 5]; average intakes of 0, 6.6, 26.7 or 107.5 mg/kg/day in males and 0, 7.7, 30.0 or 124.8 mg/kg/day in females.
Purity: About 73% isomeric mixture of [Formulation 11] and 27% triphenyl phosphate as [Formulation 5].
Vehicle: Feed
Method: Rats were examined twice daily for clinical signs and mortality, weekly physical examinations (with palpation) and measurements of body weights and food consumption. Hematology, clinical chemistry and urinalyses were performed prior to testing, at mid-test and just prior to termination. At termination, all animals were subjected to gross necropsy; organ weights of adrenal, brain, heart, liver, kidney and gonads were recorded. More than 30 organs/tissues were examined for histopathology in all groups. Brain cholinesterase was measured at termination in all groups.
Results: Treatment had no significant effect on survival, food consumption, body weight gain, hematology or clinical chemistry parameters, cholinesterase values, or the incidence of gross or microscopic lesions (including reproductive organs, brain and spinal cord). At the highest dose, there were statistically significant increases in absolute and relative liver weights in males (increased 18-24%) and females (increased ~15%), relative kidney weights in males (increased 6%), and absolute and relative adrenal weights in females (increased 12-14.7%). These changes were not accompanied by histopathological lesions or changes in clinical chemistry parameters. In this study, 400 ppm (26.7 mg/kg/day for males and 30 mg/kg/day for females) was a NOAEL and 1600 ppm (107.5 mg/kg/day for males and 124.8 mg/kg/day for females) was a LOAEL for increased liver weights in both sexes and adrenal weights in females. The study is marginally acceptable because the highest dose was considerably lower than the limit dose of 1000 mg/kg/day. The study does,

however, identify target organs that might show lesions if testing were conducted at higher exposure levels.
Reference: (Ref. 59, 60, 63)

- **Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test (OPPTS Harmonized Guideline 870.3650; OECD Guideline 422)**

No relevant studies were located that followed or were similar to the guideline listed above.

Subchronic Dermal Toxicity (21/28-day or 90-day)

***Conclusion*:**

The available subchronic dermal toxicity data were judged inadequate to meet the endpoint.

Basis for Conclusion:

The available study was conducted on a substance with a Proprietary J content of less than 50%.

- **21/28-Day Dermal Toxicity (OPPTS Harmonized Guideline 870.3200 (OECD Guideline 410)**

[Formulation 2] (43% Proprietary J; see Table 1) was applied to intact and abraded skin of New Zealand White rabbits (10/sex/group) at doses levels of 10, 100, or 1,000 mg/kg/day, 5 days/week for 3 weeks (Ref. 6); controls were treated with distilled water. Treatment-related effects included skin changes at the application site (edema at 1,000 mg/kg/day in males and ≥10 mg/kg/day in females; atonia at ≥100 mg/kg/day in both sexes; desquamation at ≥10 mg/kg/day in both sexes; and fissuring at 1,000 mg/kg/day in both sexes), higher blood urea nitrogen values at 1,000 mg/kg/day in both sexes, and dose-related depression of plasma cholinesterase at ≥100 mg/kg/day, and of erythrocyte and brain cholinesterase at ≥10 mg/kg/day in both sexes. Changes in other parameters (mortality, clinical signs, body weight, hematology, clinical chemistry, organ weights, gross or microscopic lesions) were not related to treatment.

- **90-Day Dermal Toxicity (OPPTS Harmonized Guideline 870.3250; OECD Guideline 411)**

No relevant studies were located that followed or were similar to the guideline listed above.

Subchronic Inhalation Toxicity (90-day)

Conclusion:

No available subchronic inhalation toxicity data.

Basis for Conclusion:

No pertinent studies were located that followed or were similar to the guideline listed below, or were otherwise relevant to subchronic inhalation toxicity.

- **90-Day Inhalation Toxicity (OPPTS Harmonized Guideline 870.3465; OECD Guideline 413)**

REPRODUCTIVE TOXICITY

Conclusion:

The available reproductive toxicity data were judged inadequate to meet the endpoint.

Basis for Conclusion:

No studies were located that followed or were similar to the three guidelines listed below. No histopathology of the male (testes, epididymides, prostate) or female (ovary, uterus, cervix, vagina) reproductive organs was observed in rats fed diets containing up to 1600 ppm [Formulation 5] (73% [Formulation 11]; see Note e to Table 1) for 3 months (Ref. 59, 60, 63).

- **Reproduction/Developmental Toxicity Screening (OPPTS Harmonized Guideline 870.3550; OECD Guideline 421)**
- **Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test (OPPTS Harmonized Guideline 870.3650; OECD Guideline 422)**
- **Reproduction and Fertility Effects (OPPTS Harmonized Guideline 870.3800; OECD Guideline 416)**

DEVELOPMENTAL TOXICITY

Conclusion:

The available developmental toxicity data were judged inadequate to meet the endpoint.

Basis for Conclusion:

The only available developmental toxicity data are for formulations containing only about 43% Proprietary J.

Prenatal Developmental Toxicity Study (OPPTS Harmonized Guideline 870.3700; OECD Guideline 414)

Ref. 32, 33 evaluated the developmental toxicity of [Formulation 2] (43% Proprietary J, see Table 1) in rats. In a pilot study (Ref. 32), pregnant CD rats (5/group) received undiluted [Formulation 2] at doses of 250, 500, 1,000, 2,500, or 5,000 mg/kg/day by oral gavage on gestational days (GD) 6-19; controls received 4.2 mL of water per day. Treatment had no effect on survival, behavior, or maternal necropsy findings. Anogenital staining was observed in all test groups (incidences of 1/5, 2/5, 2/5, 5/5, and 4/5 in the lowest-to-highest dose groups, respectively) and red and/or brown matter around the nose, mouth, and forelimbs in all receiving 5,000 mg/kg/day. Dose-related reductions in body weight gain for GD 0-20 were observed at $\geq$1,000 mg/kg/day, but were only biologically significant at the highest dose (-33% compared to control). At the highest dose, decreases in viable fetuses and increases in mean postimplantation losses compared to historical controls were observed at 5,000 mg/kg/day (values for concurrent controls were higher than historical control value).

In the main study (Ref. 33), groups of 25 pregnant CD rats received 2.542 mL of water or undiluted [Formulation 2] at doses of 300, 100, or 3,000 mg/kg/day by oral gavage on GD 6-19. Treatment had no significant effect on maternal survival, behavior, body weight gain, the incidence of gross necropsy findings, or most reproductive/developmental parameters (mean number of viable fetuses, postimplantation loss, early or late resorptions, total implantations, corpora lutea, fetal sex distribution, mean fetal body weight, and incidences of external malformations and visceral variations). High-dose dams had a higher incidence of clinical signs (all with yellow staining of anogenital area and half with dried red matter on nose and forepaws) compared to other groups. At 3,000 mg/kg/day, there was a slight increase compared to controls in the percentage of litters with skeletal malformations (3/24 or12% vs 1/20 or 5%); the percentage in historical controls was 6.23%

No relevant studies were located that followed or were similar to the two tests listed below.

- **Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test (OPPTS Harmonized Guideline 870.3650; OECD Guideline 422)**
- **Reproduction/Developmental Toxicity Screening (OPPTS Harmonized Guideline 870.3550; OECD Guideline 421)**

CHRONIC TOXICITY

Conclusion:

No available chronic toxicity data.

Basis for Conclusion:

No pertinent studies following or similar to the guidelines listed below or otherwise relevant to chronic toxicity were located.

- **Chronic Toxicity (OPPTS Harmonized Guideline 870.4100; OECD Guideline 452)**
- **Combined Chronic Toxicity/Carcinogenicity (OPPTS Harmonized Guideline 870.4300; OECD Guideline 453)**

CARCINOGENICITY

Conclusion:

No available carcinogenicity data.

Basis for Conclusion:

No pertinent studies following or similar to the guidelines listed below or otherwise relevant to carcinogenicity were located.

- **Carcinogenicity (OPPTS Harmonized Guideline 870.4200; OECD Guideline 451)**
- **Combined Chronic Toxicity/Carcinogenicity (OPPTS Harmonized Guideline 870.4300; OECD Guideline 453)**

NEUROTOXICITY

Conclusion:

No available neurotoxicity data.

Basis for Conclusion:

No studies examined developmental neurotoxicity or neurological function. Acceptable negative are available for delayed neurotoxicity in adults. In the 90-day oral toxicity assay by (Ref. 59, 60, 63) described above, rats exposed to [Formulation 5] (73% Formulation 11]; see Note e to Table 1) in the diet at concentrations up to 1600 ppm exhibited no neurohistopathology and no inhibition of brain cholinesterase activity.

Delayed Neurotoxicity

Conclusion:

The available delayed neurotoxicity data were judged adequate to meet the endpoint.

Basis for Conclusion:

The available studies were consistent with guidelines and overall suggest that exposure to large doses of Proprietary J does not evoke delayed neurotoxicity in hens

- **Acute and 28-Day Delayed Neurotoxicity of Organophosphorus Substances (OPPTS Harmonized Guideline 870.6100; OECD Guideline 418, 419)**

Critical Studies

Type: Acute oral delayed neurotoxicity
Species, strain, sex, number: Hen, "hybrid", females, older than 14 months, 4 water controls and 8 treated with Proprietary J
Purity: Not reported; >99% Proprietary J as [Formulation 3] (see Table 1)
Doses: The plan was 1,000 mg/kg, 5 times daily for 5 consecutive days (total 25,000 mg/kg); because of shortage of material, only 3 doses were given on day 4, so 7 doses were given on day 5 to make up the difference.
Vehicle: None
Positive control: None
Route: Oral gavage
Exposure duration, frequency: 5 days, 5 times daily
Method: Observations for up to 32 days. Birds examined daily for signs of neurotoxicity; body weights, food consumption, egg numbers, and egg weights recorded twice weekly.
Results: There were no signs of ataxia. Mortality rates, body weight gain, and feed consumption did not differ between test and control groups; egg production in test group was about 50% of controls although egg weights were slightly higher in test animals.
Reference: Ref. 28

Type: Acute oral delayed neurotoxicity
Species, strain, sex, number: Chicken, White Leghorn, females, 9/group
Purity: near pure Proprietary J
Doses: 10,000 mg/kg, twice daily (20 mg/kg/day)
Vehicle: None
Positive control: Corn oil
Route: Oral gavage
Exposure duration, frequency: Twice daily for three consecutive days; dosing regimen repeated 21 days later.
Method: Daily observations for mortality and neurotoxicity for up to 42 days. Body weights recorded at 0, 21, and 42 days. At gross necropsy, brain, spinal cord, and sciatic nerve were examined for histopathology. The neurotoxicity study was preceded by an acute oral toxicity assay in hens given 10,000 mg/kg.
Results: There were no signs of ataxia or neurohistopathological lesions in 9 hens treated with Proprietary J at cumulative doses of 120,000 mg/kg. Other [Chemical Class 3] chemicals tested at the same time were neurotoxic. The positive control, tri-*ortho*-cresyl phosphate (TOCP), caused

neurotoxicity in 4/4 hens treated with 300 mg/kg/day for 5 days (cumulative dose of 1,500 mg/kg). The acute oral LD50 in hens exceeded 10,000 mg/kg (no mortality data reported).
Reference: Ref. 34

Type: Acute oral delayed neurotoxicity
Species, strain, sex, number: Hen, female, older than 14 months, 4 control and 8 test birds
Purity: Not reported; [Formulation 3] is >99% Proprietary J and <1% stabilizers (Ref. 7)
Doses: 1,000 mg/kg five times daily for five consecutive days (= 25,000 mg/kg total)
Vehicle: None
Positive control: None
Negative control: Water
Route: Oral gavage
Exposure duration, frequency: five days, five times daily
Method: Observations for up to 32 days. Birds examined daily for signs of neurotoxicity; body weights, food consumption, egg numbers, and egg weights recorded twice weekly.
Results: No ataxia observed; no treatment-related effects on mortality or body weight gain. Mean daily food consumption in test animals was about 15% lower than in controls, largely because intake was reduced by 47% during days 0-4. In test group, egg production was about 70% of controls and egg weights about 11% lower than in controls.
Reference: Ref. 30

Additional Studies:

Several components of the [Formulation 6] series of flame retardants were isolated to >99% purity and tested at doses as high as 1,000 mg/kg in mature hens for neurotoxicity and suppression of neurotoxic esterase (Ref. 24). Details of these studies were not located. [Chemical 2], [Chemical 1], and Proprietary J elicited no signs of neurotoxicity and no suppression of NTE levels. [Chemical 7] was also judged to be non-neurotoxic, eliciting no ataxia or other signs of neurotoxicity and insignificant suppression of NTE (-4% or -15%) in two tests. However, [Chemical 8] was neurotoxic, eliciting ataxia and neurotoxicity, as well as suppression of NTE levels (by -71% and -57 to -62% in two tests) at 1 mL/kg. The author suggested that neurotoxicity was associated with [Chemical Class 1] with an oxidizable alpha-hydrogen.

Hens given [Formulation 4] (100% [Chemical Class 1]; 30-35% Proprietary J, see Table 1) at a dose of 5,000 mg/kg/day on five consecutive days by oral gavage lost weight and developed paralysis (4/4) and 3/4 died before the end of the test (Ref. 14). The study authors suggested that residual [Chemical Class 4] chemicals may have been responsible for the observed neurotoxicity.

Hens treated with 2,000 mg/kg of [Formulation 10] did not elicit clinical signs of neurotoxicity, lesions of the nervous system, or depression in NTE, whereas hens treated with TOCP at 500 mg/kg showed all of these effects (Ref. 35 abstract as described in Ref. 61).

There was no effect on survival or walking behavior among a group of 15 hens (12-14 months old) given oral doses of 11, 679 mg/kg [Formulation 5] (see Note e in Table 1) on days 1 and 21 under

EPA proposed guidelines (Ref. 58). All showed slight motor incoordination on day 1; body weights were reduced on day 38, but terminal weights were as in corn oil controls. Neurohistopathology and feed consumption were equivalent to corn oil controls. Hens treated with TOCP had increased mortality, progressive leg weakness, persistently reduced feed intake and body weight loss, and significant axonal degeneration.

No neurotoxicity studies were located that were relevant to the categories listed below.

Neurotoxicity (Adult)

- **Neurotoxicity Screening Battery (OPPTS Harmonized Guideline 870.6200; OECD Guideline 424)**

Developmental Neurotoxicity

- **Developmental Neurotoxicity: Developmental Neurotoxicity Study (OPPTS Harmonized Guideline 870.6300)**

Additional neurotoxicity studies:

- Schedule-Controlled Operant Behavior (mouse or rat); OPPTS Harmonized Guideline 870.6500
- Peripheral Nerve Function (rodent); OPPTS Harmonized Guideline 870.6850
- Sensory Evoked Potentials (rat, pigmented strain preferred); OPPTS Harmonized Guideline 870.6855

These additional neurotoxicity studies may be indicated, for example, to follow up neurotoxic signs seen in other studies, or because of structural similarity of the substance to neurotoxicants that affect these endpoints. These studies may be combined with other toxicity studies.

Other Neurotoxicity Data

Cholinesterase inhibition

In five hens that received 25,000 mg/kg of [Formulation 7] in divided doses (8, 8, and 9 g/kg at 4-hour intervals) by oral gavage, plasma cholinesterase (pChE) levels 30-60 minutes later were about 60-70% of pre-dose levels (Ref. 29); 9 days later, pChE levels had risen to 83.1% of the pre-dose level. One bird that showed clinical signs (quiet with subdued behavior) after dosing did not show appreciable recovery of pChE on day 9. The authors concluded that because of the very high dose administered, the test material was not a significant inhibitor of plasma cholinesterase.

IMMUNOTOXICITY

Conclusion:

No available immunotoxicity data.

Basis for Conclusion:

No pertinent studies were located that followed or were similar to the guideline listed below, or were otherwise relevant to immunotoxicity.

- **Immunotoxicity (OPPTS Harmonized Guideline 870.7800)**

GENOTOXICITY

Conclusion:

The available genotoxicity data were judged inadequate to meet the endpoint.

Basis for Conclusion:

The available studies followed methods equivalent to guidelines, but tested materials for which the Proprietary J content was low (less than 50%) or uncertain; the latter studies were only available as robust summaries. None of the studies indicate the Proprietary J-containing mixtures are mutagenic in bacteria or mammalian cells.

Gene Mutation in Vitro:

- **Bacterial Reverse Mutation test (OPPTS Harmonized Guideline 870.5100; OECD Guideline 471)**

[Formulation 1] (typical analysis 43% Proprietary J; see Table 1) was not mutagenic in *S. typhimurium* strains TA98, TA100, TA1535, TA1537, or TA1538 with or without metabolic activation (Ref. 15).

[Formulation 2] (typical analysis 43.2% Proprietary J, see Table 1) at concentrations between 0.01 and 10 μL/plate produced negative results in *S. typhimurium* strains TA98, TA100, TA1535, TA1537, or TA1538 and *Saccharomyces cerevesiae* D4 with or without metabolic activation (Ref. 36).

As described in a robust summary, [Formulation 5] (see Note e in Table 1) at concentrations between 0.005 and 10 μg/plate produced negative results in *S. typhimurium* strains TA98, TA100, TA1535, TA1537, or TA1538 with or without metabolic activation (Ref. 38); cytotoxicity was observed at 0.1 μg/plate and higher.

- **In vitro Mammalian Cell Gene Mutation Test (OPPTS Harmonized Guideline 870.5300; OECD Guideline 476)**

[Formulation 2] (typically 43% Proprietary J, see Table 1) at concentrations between 0.011 and 0.1 μL/mL (0.2 μL/mL was cytotoxic) did not induce forward mutations in cultured mouse lymphoma L5178Y/TK$^{+/-}$ cells with or without metabolic activation (Ref. 37).

As described in a robust summary, [Formulation 5] (see Note e in Table 1) at concentrations between 0.975 and 125 nL/mL (≥15.6 nL/mL was cytotoxic) did not induce forward mutations in cultured mouse lymphoma L5178Y/TK$^{+/-}$ cells with or without metabolic activation (Ref. 39).

Chromosomal Aberration in Vitro

- **In Vitro Mammalian Chromosome Aberration Test (OPPTS Harmonized Guideline 870.5375)**

As described in a robust summary, [Formulation 5] (see Note e in Table 1) at concentrations between 0.625 and 20 nL/mL (≥2.5 nL/mL was cytotoxic) did not increase the frequency of chromosomal aberrations in cultured mouse lymphoma L5178Y/TK$^{+/-}$ cells with or without metabolic activation (Ref. 40).

No studies were located that were relevant to the categories listed below.

Gene Mutation in Vivo
Chromosomal Aberration in Vivo
DNA Damage and Repair

Other

- **In vitro Sister Chromatid Exchange Assay (OPPTS Harmonized Guideline 870.5900)**

As described in a robust summary, [Formulation 5] (see Note e in Table 1) at concentrations between 0.625 and 20 nL/mL (≥2.5 nL/mL was cytotoxic) did not increase the frequency of sister chromatid exchanges in cultured mouse lymphoma L5178Y/TK$^{+/-}$ cells with or without metabolic activation (Ref. 40).

Ecotoxicity

Acute Toxicity to Aquatic Organisms

Conclusion:

The available acute toxicity data for fish, aquatic invertebrates, and algae were judged inadequate to meet the endpoints.

Basis for Conclusion:

Summary data were located for a screening-level assessment of the acute toxicity of Proprietary J to rainbow trout and bluegill (Ref. 31). Proprietary J was dissolved in acetone and added to water. Fish were exposed to aqueous solutions of 0.1, 1.0, 10.0, or 100 mg Proprietary J/L for 96 hours. The estimated 96-hour LC_{50} values for rainbow trout and bluegill were 1.1 and 1.0 mg/L, respectively. The two highest test concentrations exceeded the reported water solubility of Proprietary J (Ref. 49). The summary did not provide sufficient information regarding study conditions, including test substance purity, to allow for an independent evaluation of the studies.

Summary data were located for studies of the toxicity of Proprietary J to *Daphnia magna* and the midge *Chironomus tentans* (Ref. 62). The reported 48-hour LC_{50} values for *D. magna* and *C. tentans* were 0.30 and 0.15 mg/L, respectively. Study details, including test substance purity, were not presented in the summary, so the results could not be independently evaluated.

Summaries were located for studies of the acute toxicity of the commercial aryl phosphate ester mixtures [Formulation 8], [Formulation 9], and [Formulation 2] to fish, aquatic invertebrates, and algae (revised HPV Robust Summaries submitted by Ref. 2, as part of the HPV Challenge Program). Although some of the tested products may have contained Proprietary J, their actual composition was not presented in the study summaries. Without precise knowledge of the composition of the tested materials, it is not possible to use these studies to make a definitive statement regarding the toxicity of Proprietary J. [Formulation 2] has been reported (by a different chemical company) to contain <50% Proprietary J (Table 1).

Studies of the acute toxicity of [Formulation 8] to rainbow trout, bluegill, fathead minnow, channel catfish (Ref. 8), *D. magna*, the midge *C. plumosus*, the amphipod *Gammarus pseudolimnaeus*, and algae (Ref. 50) were located. [Formulation 8] contains 15-20% [Chemical 3] and unspecified amounts of at least five other compounds (Ref. 8). Chemical analysis of the aqueous test solutions in a chronic toxicity study (discussed below) (Ref. 8) suggested that the concentration of Proprietary J in the test waters may have been 40% or less of nominal concentrations of [Formulation 8]. Given that the organisms in these tests were exposed to a mixture of compounds, which was predominantly not Proprietary J, it is concluded that it is not possible to use these studies to make a definitive statement regarding the toxicity of Proprietary J.

No additional, pertinent acute toxicity studies with fish, aquatic invertebrates, or algae were located that addressed the endpoints in the guidelines listed below.

- **Acute Toxicity to Freshwater and Marine Fish (OPPTS Harmonized Guideline 850.1075; OECD Guideline 203)**
- **Acute Toxicity to Freshwater Invertebrates (OPPTS Harmonized Guideline 850.1010; OECD Guideline 202)**
- **Acute Toxicity to Marine/Estuarine Invertebrates (OPPTS Harmonized Guideline 850.1035)**
- **Algal Toxicity (OPPTS Harmonized Guideline 850.5400; OECD Guideline 201)**

Chronic Toxicity to Aquatic Organisms

Conclusion:

The available chronic toxicity data for fish and aquatic invertebrates were judged inadequate to meet the endpoints.

Basis for Conclusion:

Studies of the chronic toxicity of the commercial phosphate ester compound [Formulation 8] to fathead minnow (Ref. 8), *Daphnia magna*, the midge *Chironomus plumosus*, and the amphipod *Gammarus pseudolimnaeus* (Ref. 50) were located. Formulation 8] contains 15-20% [Chemical 3] (Ref. 8). Measured concentrations of Proprietary J in the test waters were 25% to 40% of nominal concentrations of [Formulation 8] (Ref. 8). Thus, the organisms in these tests were exposed to aqueous solutions that contained a mixture of compounds that were predominantly not Proprietary J. Therefore, it is concluded that it is not possible to use these studies to make a definitive statement regarding the toxicity of Proprietary J.

No additional, pertinent chronic toxicity studies with fish or aquatic invertebrates were located that addressed the endpoints in the guidelines listed below.

- **Chronic Toxicity to Freshwater and Marine Fish (OPPTS Harmonized Guideline 850.1400; OECD Guideline 210)**
- **Chronic Toxicity to Freshwater Invertebrates (OPPTS Harmonized Guideline 850.1300; OECD Guideline 211)**
- **Chronic Toxicity to Marine/Estuarine Invertebrates (OPPTS Harmonized Guideline 850.1350)**

Acute and Subchronic Toxicity to Terrestrial Organisms

Conclusion:

No available acute and subchronic toxicity data for terrestrial organisms.

Basis for Conclusion:

No pertinent acute oral, acute dietary, or reproductive toxicity studies with birds and no subchronic toxicity studies with earthworms were located that addressed the endpoints in the guidelines listed below.

- **Acute Oral Toxicity in Birds (OPPTS Harmonized Guideline 850.2100)**
- **Acute Dietary Toxicity in Birds (OPPTS Harmonized Guideline 850.2200; OECD Guideline 205)**
- **Reproductive Toxicity in Birds (OPPTS Harmonized Guideline 850.2300; OECD Guideline 206)**
- **Earthworm Subchronic Toxicity (OPPTS Harmonized Guideline 850.6200; OECD Guideline 207)**

Physical/Chemical Properties

Proprietary J: Aryl phosphate
CAS
MF
MW
SMILES

Water Solubility (mg/L):
Conclusion: The available water solubility data are adequate.
Basis for Conclusion: Ref. 49 gives a measured value for the water solubility of Proprietary J.

Solubility (mg/L)	References
3.20	Ref. 49, 51

Log K_{ow}:
Conclusion: The data for this endpoint are adequate.
Basis of Conclusion: The Log K_{ow} values of 5.12 are identical as found in two sources Ref. 49 and Ref. 3). Ref. 4 estimates the Log K_{ow} value based on HPLC that are in reasonable agreement with the key study indicated above. Ref. 45, however, gives a Log K_{ow} value of 13.2, which is much higher than that found in the other sources and this value does not appear reasonable for compounds of this class.

Log K_{ow}	Reference
5.12	Ref. 3, 49, 51
3.23 4.76 6.44	Ref. 4 (estimated from reverse phase HPLC data by Ref. 48)
13.2	Ref. 45
13.3	Ref. 13

Oxidation/Reduction: No data

Melting Point:
Conclusion: The data are adequate for this endpoint.
Basis for Conclusion: A value of -20 ˚C is given for the melting point of Proprietary J. Another study provides a pour point for Proprietary J that is in reasonable agreement with the melting point value.

Melting Point (˚C)	Reference
-20	Ref. 13
-21 (pour point)	Ref. 45

Boiling Point:
Conclusion: The available boiling point data are adequate to characterize this endpoint.
Basis for Conclusion: Two sources contained measured boiling point information. A third source required calculating the boiling point from the Clausius-Clapeyron equation using data measured by Ref. 9. The boiling points given here (including the calculated boiling point) are within a reasonable range of each other.

BP (˚C/torr)	References
261/6	Ref. 49, 51
425/760 dec. 405	Ref. 9 (extrapolated according to the Clausius-Clapeyron Equation using experimentally-derived parameters: Log P(torr) = -A/T + C, where T is in Kelvin, A= 4444, C=9.24) The decomposition temperature was reported in this same paper.
420/760	Ref. 4 (estimated using Meissner's method)
155/2	Ref. 13

Vapor Pressure (torr):
Conclusion: The majority of available vapor pressure data give an adequate endpoint.
Basis for Conclusion: Two sources contained measured vapor pressure data. These two values were in agreement with each other. The third source required calculating the boiling point from the Clausius-Clapeyron equation using data measured by Ref. 9. The calculated value was in reasonable agreement with the measured values.

VP (torr/˚C)	Reference
1.40×10^{-6}/25	Ref. 51; Ref. 4 (measured)
4.6×10^{-7}	Ref. 4 (estimated from b.p. using Method 2 in Ref. 21)
2.16×10^{-6}/25	Ref. 9 (extrapolated according to the Clausius-Clapeyron Equation using experimentally-derived parameters Log P(torr) = -A/T + C, where T is in Kelvin, A= 4444, C=9.24)
1.35/200	Ref. 45
10.2/250	Ref. 13

Odor: No data

Oxidation/Reduction Chemical Incompatibility: No data

Flammability: No data

Explosivity: No data

Corrosion characteristics: No data

pH: No data

UV/VIS absorption: No data

Viscosity:
Conclusion: The viscosity of this compound has been adequately characterized.
Basis for Conclusion: A single study on the viscosity of Proprietary J was located and appears reasonable given the other physical/chemical properties available for this compound.

Viscosity at 25 ˚C	**Reference**
58	Ref. 13

Density/Relative Density/Bulk Density:
Conclusion: The density of this compound has been adequately characterized.
Basis for Conclusion: A single study on the density of Proprietary J was located and appears reasonable given the other physical/chemical properties available for this compound.

Density	**Reference**
1.175-1.185	Ref. 13

Dissociation Constant in Water: No data

Henry's Law Constant:
Conclusion: The data are adequate to characterize the Henry's Law constant.
Basis for Conclusion: The key study provides an estimated Henry's Law constant based on measured vapor pressure and water solubility data and is taken from Ref. 45. This is a reasonable method for estimating a Henry's Law constant. The other studies identified provide estimates although they are based on estimated vapor pressures and not experimental values and are not sufficiently reliable to categorize this end point.

Henry's Law Constant	Reference
8.48×10^{-7} atm-m^3/mole	Ref. 45, 51
7.2×10^{-8} atm-m^3/mole	Ref. 4 (calculated from vapor pressure of 4.6×10^{-7} mm Hg and water solubility 3.2)
2.2×10^{-7} atm-m^3/mole	Ref. 4 (calculated from vapor pressure of 1.4×10^{-6} mm Hg and water solubility 3.2)
2.15×10^{-5} atm-m^3/mole	Ref. 46

Environmental Fate

Bioconcentration

Fish:

Conclusion: The bioconcentration of Proprietary J has not been adequately characterized.

Basis for Conclusion: Studies conducted by Ref. 44 give three different methods for determining the BCF value for both rainbow trout and fathead minnows. The first two methods used were Biofac and initial rate. Biofac is a computer program that requires constant water concentration for its calculations. The "initial rate" method assumes that the rate of uptake of Proprietary J from water is much greater than the rate of clearance during the initial exposure period. The static test method yields equilibrium BCFs if the fish exposure continues until a maximum concentration is observed. The maximum concentration was not reached in rainbow trout, so the BCFs may be underestimated. Although the maximum concentrations appeared to have been reached in fathead minnows, further studies conducted by Ref. 46 show the BCF value for fathead minnows measured using the static test method may be very different to those measured previously (Ref. 44) using this same method. Given that none of these tests were conducted according to EPA or OECD guidelines, and that the results vary widely, this endpoint is not adequately characterized by the available experimental data.

Reference	Species	BCF	Key Design Parameters				Comments
			Exp. type	Range (ppb)	Study length	T (˚C)	
Ref. 44	Rainbow trout	1096	Static	1.8-55	1-24 hours	10	The calculation of BCF from this method comes from the following equation: $k_1=[CFish(max)k_2]/[A \exp(-Btmax)]$ and is based on the total ^{14}C.
Ref. 44	Rainbow trout	1335	Biofac	1.8-55	1-24 hours	10	k_1 and k_2 values were estimated by use of this nonlinear regression program. In these calculations, the initial exposure concentration (0 hr) was used.
Ref. 44	Rainbow trout	2298	Initial rate	1.8-55	1-24 hours	10	The calculation of BCF from this method comes from the following equation: $k_1=(\Delta CFish/\Delta t)Cw$

Reference	Species	BCF	Key Design Parameters				Comments
			Exp. type	Range (ppb)	Study length	T (˚C)	
Ref. 44	Fathead minnow	498	Biofac	0.8-36.5	1-24 hours	10	k_1 and k_2 values were estimated by use of this nonlinear regression program. In these calculations, the initial exposure concentration (0 hours) was used.
Ref. 44	Fathead minnow	785	Static	0.8-36.5	1-24 hours	10	The calculation of BCF from this method comes from the following equation: $k_1=[CFish(max)k_2]/[A exp(-Btmax)]$
Ref. 44	Fathead minnow	3316	Initial rate	0.8-36.5	1-24 hours	10	The calculation of BCF from this method comes from the following equation: $k_1=(\Delta CFish/\Delta t)Cw$
Ref. 45	Rainbow trout	1096	Static	5-50	24 hours		Ref. 45 takes this value from Ref. 44.
Ref. 45	Fathead minnow	785	Static	5-50	24 hours		Ref. 45 takes this value from Ref. 44.
Ref. 46	Fathead minnow	528		50	8 hours		
Ref. 4	Estimated	4400					The value is estimated from measured values of Log K_{ow} from Ref. 49 and Ref. 41 using the equation in Ref. 5).

Daphnids: No data

Green Algae: No data

Oysters: No data

Earthworms: No data

Fish Metabolism: No data

Degradation and Transport

Photolysis in the Atmosphere: No data

Photolysis in Water:
Conclusion: This endpoint has been adequately characterized.
Basis for Conclusion: The products from the photolysis of Proprietary J in water as determined by GC/MS have been summarized (Ref. 13). The products are [Chemical 9] and [Chemical 10].

Photolysis in Soil: No data

Aerobic Biodegradation:
Conclusion: The biodegradation of Proprietary J under aerobic conditions has not been adequately characterized.
***Basis for Conclusion*:** Several different types of studies have been carried out and the weight of evidence indicates that Proprietary J is likely to biodegrade under aerobic conditions. Only the studies by Ref. 23 differ greatly from those in other literature sources, which is likely a result of the water/sediment innoculum used.

Study type/ Method	Innoculum	Acclim	Degradation	Time	Comments	Reference
Thompson-Duthie-Sturm Procedure	Activated sludge		90% as CO_2 Evolution	28 days		Ref. 43
Monsanto Shake Flask Procedure	Activated sludge		43% as CO_2 Evolution	28 days		Ref. 43
River Die-away		4 days	50%	11 days	The initial concentration was 1 ppm; after 4 days, 50% primary degradation occurred.	Ref. 43, 49
Simulated Biological Treatment/ SCAS	Activated sludge		93+ 84+/-3	9 weeks 8 weeks	3 mg/L/24 hours 13 mg/L/24 hours	Ref. 13, 49
	Activated sludge	24-hour cycle	>93 84+/-3	1 day	3 ppm/cycle 13ppm/cycle	Ref. 43

Study type/ Method	Innoculum	Acclim	Degradation	Time	Comments	Reference
SCAS and RDA Analytical Method (Method AC-72-M-S)	Activated sludge with domestic sewage feed		75.9% recovery	24 hours	Mixed liquor extraction	Ref. 43

Anaerobic Biodegradation: No data

Porous Pot Test: No data

Pyrolysis: No data

Hydrolysis as a Function of pH: No data

Sediment/Water Biodegradation:
Conclusion: The biodegradation of Proprietary J in the presence of pond and/or river sediment has been adequately characterized.
Basis for Conclusion: Biodegradation of Proprietary J has been studied under a variety of conditions and temperatures in the presence of both river and pond sediment. The weight of evidence indicates the potential for Proprietary J to degrade under these environmental conditions.

Sediment	Temp.	$T_{1/2}$	Comments	Reference
Pond water	25	0.44 days	Time interval was 0-3 days.	Ref. 46
Pond sediment	25	39	Static conditions. Sediment was collected from a pond made specifically for this experiment at Glenlea Research Station, University of Manitoba. Initial Proprietary J concentration 0.10 µg/mL. Sediment:water ratio 1:10. Time interval was 2-105 days.	Ref. 46
Pond sediment	25	4.2	Time interval was 0-6 days.	Ref. 47
Pond sediment	10	5.5	Time interval was 0-6 days.	Ref. 47
Sediment-water microcosms			39% (0.1 mg concentration), 18% (1 mg concentration), and 5% (10 mg concentration) mineralization after 8 weeks with innoculum from Lake Chicot, AR after 1 week of lag time.	Ref. 23

Sediment	Temp.	$T_{1/2}$	Comments	Reference
Sediment-water microcosms			14% (0.1 mg concentration), 8% (1 mg concentration), and 2% (10 mg concentration) mineralization after 8 weeks with innoculum from Little Dixie Reservoir, MO after 1 week of lag time.	Ref. 23
Sediment-water microcosms			12.5% (0.1 mg concentration and 10 mg concentration) mineralization after 8 weeks with innoculum from Redfish Bay, TX.	Ref. 23
Sediment-water microcosms			1.9% (0.1 mg concentration) and 1.8% (10 mg concentration) mineralization after 8 weeks with innoculum DeGray Reservior, AR.	Ref. 23
Sediment-water microcosms			9.9% (0.1 mg concentration) and 6% (10 mg concentration) mineralization after 8 weeks with innoculum Arkansas River, AR.	Ref. 23
Pond sediment	5	16.1	Time interval was 0-6 days.	Ref. 47

Soil Biodegradation with Product Identification: No data

Indirect Photolysis in Water: No data

Sediment/Soil Adsorption/Desorption:
Conclusion: The K_{oc} has not been adequately characterized.
Basis for Conclusion: In both literature reports (Ref. 45 and Ref. 4), the values obtained for the K_{oc} are calculated. No experimental values for this endpoint were located.

K_{oc}	Source	Reference
14600	Calculated from K_{ow} using the Kenaga and Goring equation	Ref. 45
2300	Calculated from water solubility using the Kenaga and Goring equation	Ref. 4